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HEART FAILURE

PART II

THE DIASTOLIC VOLUME LAW AND FAILURE

The fundamental energetic fact in connection with the adaptability of the heart to its load is the diastolic volume law.[28] This law states that when chemical conditions and heart rate are held constant the energy output of the heart in contraction is a function of the length of its fibers at the end of diastole (that is at the initiation of contraction). Furthermore, it has long been known[29] that the conditions which call for an increase in cardiac work, (a) increased venous filling and (b) increased arterial pressure, both increase the diastolic cardiac volume. Increased filling does so for obvious reasons; increased arterial pressure does so because the residual blood after systole increases with each contraction as long as the energy output in systole is insufficient to move the blood entering in diastole. The process of accumulation ceases when the diastolic ventricular volume rises to a level such that the energy liberated is adequate to carry the load of work imposed.

The law of diastolic volume is also one of the most basic physiological considerations in heart failure. The entire symptomatology of congestive failure can be accounted for on the basis of the diastolic volume law. In this consideration the presence or absence of hypertrophy will be ignored because it has no influence upon the basic process. Hypertrophy only delays onset of the symptoms because it makes the heart a stronger pump-engine.

When a heart fails it dilates. This dilation is the result of an increase in residual blood. The ventricles are unable to eject in systole all that enters in diastole. This process is slow and over any short period of time the heart is in a steady state because the dilation results in a greater systolic energy liberation which permits it to carry its load.

Congestive failure is progressive.[30] The intimate reasons for its onset are unknown. The result is a decrease in mechanical efficiency. The cause may be the exhaustion of some important metabolite, coenzyme, enzyme or structural element, or the accumulation of some inhibitor. This question richly warrants intensive study because it is at the bottom of the whole clinical problem. But the progressive character of the condition is a fact of universal experience. It results in a progressively greater increase in residual blood and consequently in the volume of the heart.

The dilation of the heart results from the pressure of blood in the great veins. Since, as noted above, the heart is an elastic bag, its internal pressure must rise if it is to be distended. Since it shows hysteresis it adapts somewhat to this pressure, but in the end the pressure necessary to distend it increases considerably. The venous pressure therefore rises because the return flow of blood to the great veins occurs normally under a large pressure gradient. Normally the pressure in the right atrium in diastole is from 10 to 15 mm. Hg. lower than the pressures in small venules. This difference of pressure drives the blood to the heart. When the heart fails the atrial pressure rises toward the level of the venule pressure. The pressure in the great veins is a resultant of two factors: pressure in the small veins and the rate at which blood can leave the great veins and enter the chambers of the heart. When the latter are fuller than normal in protodiastole, pressure in the great veins inevitably rises and tends to approach more closely the pressure in small veins because the normal rate of run-off into the heart does not prevail.

The account just given of the hemodynamics involved in elevation of the venous pressure in cases of heart failure is a detailed statement of the so-called backward failure mechanism. This title emphasizes the effects of the increased cardiac residual blood volume upon the dynamics of the symptom complex of heart failure.

Backward failure is the antithesis of forward failure, in which, as in ventricular fibrillation, the heart simply fails to propel blood. In the presence of myocardial ischemia there may be varying proportions of the symptoms due respectively to the back-pressure effects and to the failure to maintain adequate forward movement of blood. Always terminally it is the failure to maintain forward movement that produces death, but the predominating symptoms before the terminal state are usually due to the back-pressure.

THE ROLE OF THE CORONARY BLOOD FLOW IN HEART FAILURE

When portions of the myocardium are deprived of blood, such heart muscle unquestionably fails by virtue of decreased, and ultimately of absence of energy liberation. Coronary arterial occlusion stops the supply of oxygen to the myocardium, and any part of the heart so affected rapidly loses its contractile function. Likewise obstruction to coronary venous outflow impedes the intracardiac circulation. Increases in right atrial, or right intraventricular, pressures, or both, measurably quicken the process of failure in experimental animals[18] and presumably in man. Thus, elevations in venous pressure set up a vicious cycle—decreasing coronary flow and thus aggravating failure, which in turn results in still higher venous pressures, perpetuating the cycle. It may be because this cycle is interrupted that reduction in volume of circulating blood is sometimes

therapeutically effective[31] in treatment of congestive failure.

The rate of blood flow in the coronary arteries is dependent upon the pressure gradient between the stomata of the coronary arteries in the aortic wall and the effluents of the coronary and thebesian veins. Thus the difference between aortic pressures on the one hand and right atrial and intraventricular pressures on the other, chiefly determines the coronary flow. This difference can be altered by changes on either side.[18] Consequently a rise in aortic pressure can compensate for the ill-effects of elevations in right heart pressure. It is well known that a fall in arterial pressure quickly embarrasses the heart by reduction in coronary flow. The effect of increased arterial pressure has not been studied critically but it has been suggested[32] that in some cases in which mitral stenosis has been present, the supervention of hypertension has postponed the development of symptoms of decompensation.

SUMMARY

The heart is a pump-engine which ordinarily fails by virtue of a decline in mechanical efficiency of unknown cause. The result of the efficiency decline is an increase in systolic ventricular volume in consequence of the obedience of the heart, even in failure, to the diastolic volume law. As a consequence of the diastolic volume law the decompensated heart is enlarged, the pressure in the great veins rises, while the arterial blood pressure and output of the heart at rest are little if at all affected. The cardioactive glycosides increase the mechanical efficiency of the decompensated heart. This action appears to be the basic mechanism by which such agents alleviate the symptoms of cardiac failure. The flow of coronary blood is impeded by elevations in right heart pressures and by low aortic pressures. The elevation in right heart pressure in the presence of failure sets up a vicious cycle which tends to make failure a progressive phenomenon. The rational approach to the understanding of heart failure is through consideration of the heart as a machine, the energetics of which can be analyzed and influenced in the direction of more effective function.

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